AN ABSTRACT OF THE THESIS OF

John William Burnham for the degree of Master of Science in Environmental Health Management presented on March 6, 1996.

Title: Lead Exposures in Radiator Shops in a Nine-County Area of Northwestern Oregon.

Redacted for Privacy

Abstract approved:

Annette M. Rossignol

The primary objective of this study was to assess radiator repair shops' compliance with Oregon OSHA Lead and Hazard Communication regulations. Fifty radiator repair shops were randomly selected from nine counties that comprise the highest population density in Oregon. All 50 worksites were visited; of these 50 worksites, 22 were within the scope of the emphasis program and were inspected. Personal monitoring for lead exposure was performed in 19 of the 22 inspected sites. A total of 151 violations were cited during the 22 inspections, including 85 violations of the Lead Standard and 44 violations of the Hazard Communication Standard. Citations were issued at 21 of the 22 worksites. Thirty-two airborne lead exposures were measured for individual workers at the 19 worksites where air monitoring was performed (one worker used lead-free solder). Personal airborne lead exposures ranged from 6.2 to 236.8 micrograms/cubic meter. Mean airborne lead exposure was 76.7 micrograms/cubic meter. Core header

cleaning appeared to be responsible for the high-end exposures. Ventilation approaches used by the lead monitored shops included general, canopy, capture and enclosure. Blood lead records at five of the lead-monitored shops revealed that blood lead measurements had never been performed for a total of eight workers. Of the 32 workers evaluated for airborne lead exposures, only 19 had received blood lead tests in the year prior to the inspection date. The mean blood lead level for these 19 workers was 34.7 micrograms/100grams whole blood. The highest blood lead level was 61 micrograms/100grams. Airborne lead exposures in radiator repair shops largely were controlled by ventilation design and personal work habits.

Lead Exposures in Radiator Shops in a Nine-County Area of Northwestern Oregon

by

John William Burnham

A THESIS

submitted to

Oregon State University

in partial fulfillment of the requirements for the degree of

Master of Science

Completed March 6, 1996

Commencement June 1996

Master of Science thesis of John William Burnham presented on March 6, 1996

APPROVED:

Redacted for Privacy

Major Professor, representing Environmental Health Management

Redacted for Privacy

Chair of Department of Public Health

Redacted for Privacy

Dean of Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

Redacted for Privacy

John William Burnham, Author

Acknowledgment

This study was supported by the Oregon Occupational Safety and Health Division (OR-OSHA). The author also wishes to acknowledge Joy Flack, Regional Industrial Hygienist, Seattle Regional Office, Federal OSHA for contributions early in the study.

DEDICATION

This thesis is dedicated to my loving wife, Lynn, for the patience and support that made completion of this effort possible. I also dedicate this effort to Amber Grace, Amy Lynn and John Ashcroft.

TABLE OF CONTENTS

LIST OF TABLES

Lead Exposures in Radiator Shops in a Nine-County Area of Northwestern Oregon

INTRODUCTION

Lead metal usage by ancient and modern man and woman is well documented.[1] Consequently, history is replete with documented cases of lead poisoning. Hippocrates reported a case of severe colic in a metal worker in the fifth century B.C.[2] In the second century B.C., Nicander of Colophon, a Greek physician, wrote detailed descriptions of symptoms exhibited by a patient who worked with the metal.[3] Lead was used in ancient Rome for water tanks, plumbing, kitchen tools, as a food additive, and in the preparation of alcoholic drinks. Recent authors even suggested that lead may have played a significant part in the decline of the Roman Empire.[4,5] Symptoms described by ancient authors included constipation, abdominal pain, pallor, hallucinations, weight loss and paralysis.

Throughout its history, lead was used as pharmaceutical. This practice continued into the current century. Lead preparations were listed in the Textbook of Pharmacology and Therapeutics in England in 1910.[6] Later, large amounts of lead were used to treat cancer in the United States.[7]

The four routes of lead intake and absorption are: 1) respiratory tract; 2) gastrointestinal tract; 3) skin; and 4) placenta. Other factors also influence the rate of entry into the bloodstream. These factors are the amount of lead in the exposure media, the physicochemical properties of the specific lead compound, as well as the age, sex, physical condition and dietary deficiencies of the individual exposed.[8]

After absorption, lead becomes widely distributed in virtually all body fluids. However, excretion is primarily via fecal and urinary pathways. The lead excretion rate is influenced heavily by its high affinity for bone tissue. Rabinowitz recently proposed the existence of two bone subcompartments for lead deposition.[9] These two subcompartments are the spongy and cortical bone which were characterized as the "slow pool" and the "very slow pool", respectively. Experimental and epidemiological studies have demonstrated that a high proportion of absorbed lead is retained by the body.[10,11]

Biological monitoring (BM) for lead has been used extensively for prevention of exposure. Typical BM includes lead in blood, urine, bone, teeth and hair, as well as zinc - protoporphyrin (ZPP). Blood lead level (BLL) is clearly the most useful form of BM. BLL reflects the equilibrium between absorption, retention and excretion rate.[12] Two broad-based BLL studies of the non-occupationally exposed population in the United States were carried out between 1976 - 1980.[13,14] These studies reported an arithmetic mean of 13.9 micrograms per deciliter (μg/dL) and a geometric mean of 7.5 μg/dL, respectively. Other studies found statistically significantly higher (3%) BLL in men and a correlation between alcoholic beverage consumption and a 12% increase in BLL.[15,16]

Several chemical agents are capable of chelating biological lead.[17] Some of the more commonly used chelating agents include calcium disodium ethylenediamine tetraacetic acid (EDTA) and dimercaprol. Chelating agents can be of value therapeutically as well as allowing researchers to develop a clearer understanding of lead metabolism and its association with various organs and tissues.[17,18]

Calcium disodium EDTA was determined to possess a high equilibrium constant for lead compared to other common metals in the body.[19] This preferential attachment to lead coupled with rapid excretion rates in feces and urine have resulted in EDTA becoming an important pharmacological agent.[20] Toxicity effects can be reduced by using calcium disodium EDTA as opposed to the disodium EDTA salt.

Acute and chronic lead poisoning can present themselves through clinical or sub-clinical symptomatology. Lead toxicity may be expressed by a broad spectrum of physiologic systems. Those systems include the neurologic, nephrologic, erythropoietic, cardiovascular, gastrointestinal, hepatic, reproductive, endocrine and immunologic systems.[8] Animal studies label some forms of inorganic lead as a suspect carcinogen.

In adults, acute encephalopathy is relatively rare, especially in occupational settings. During the past 30 years, over 90% of the documented cases have been attributed to moonshine consumption, contaminated wine, lead-glazed ceramics and food contamination.[21]

Lower exposure levels can lead to cognitive and behavioral neurotoxicity. In exposed workers, neurobehavioral surveys often result in limited reliability of findings because of a number of difficult to examine confounding factors.[8] Ryan et al. recently concluded that workers with BLL below 40 µg/dL had no detectable neuropsychological functioning disorders.[22] This study included 288 workers exposed to lead and 181 nonexposed controls. Exposed workers had an average BLL of 40.1 µg/dL and controls averaged 7.2 µg/dl. The test battery used was the Pittsburgh Occupational Exposure Test Battery which includes 21 tests grouped into five neuropsychological functions.

Nephropathy related to lead exposure was described over a century ago.[23] Over the years, a number of acute and chronic forms of renal involvement have been identified. Tubular dysfunctions are often the consequence of acute lead poisoning. Commonly reported tubular dysfunctions include Fanconi syndrome and Wilson's disease, both often resulting in glycosuria, aminoaciduria, hyperphosphaturia and hypophosphatemia. The frequency of tubular dysfunctions appears to be higher for children than adults with BLL ranging from 50-120 µg/dL for symptomatic children.[24]

Chronic lead nephropathy has been reported for both environmental and occupational exposures. Prolonged low-dose exposure (< 25 $\mu g/m^3$) generally leads to subclinical impairments in renal function. However, Cooper et al. surveyed death certificates for lead workers in the U.S. from 1947 - 1980 and reported a significantly higher incidence of hypertensive and idiopathic renal diseases.(26)

Occupational lead exposure and gout was first reported in 1859.(27) Garrod reported that 30% of gouty patients were painters. This high proportion was confirmed later by Lancereaux.(28) Many recent studies indicate an inverse correlation between renal function and body lead burden in patients with gout.(29)

Many of the symptoms described by the ancient Greeks, such as pallor and weakness, were likely a result of the adverse effects of lead on the erythropoietic system.(3) The first report of lead-induced anemia occurred in 1831.(30) Later experimental studies established that anemia resulting from lead poisoning is a consequence of hemolysis of erythrocytes,(31) as well as interference with heme synthesis.(32) Acute exposures are generally manifested as acute hemolytic anemia.

Interference with heme synthesis is largely a result of lead inhibition of enzymes responsible for heme synthesis. One of the more prominent indicators of this interference is the formation of erythrocyte ZPP. ZPP is often determined in conjunction with direct BLL during biological monitoring.[12] BLL still is the preferred outcome from biological monitoring. Dose-effect curves have been published directly relating BLL and hemoglobin concentrations. Whitehead recently reported a predictable mathematical relationship between these two variables.[33]

The impact of lead poisoning on the erythrocytes of the erythropoietic system obviously extends to alteration of cardiovascular system function. A less obvious impact on the cardiovascular system was the discovery of an apparent relationship between lead exposure and high blood pressure,[35] although not all investigators have reported findings supporting this association.[35]

One recent study that reported a positive correlation between lead poisoning and high blood pressure involved 96 smelter workers and an equal number of controls.[36] Average BLL for the exposed group was 51 µg/dL (SD=16) while the control group average was 11 µg/dL (SD=3). Diastolic blood pressure for exposed workers averaged 5 mm Hg higher in the supine position compared to controls.

Gastrointestinal effects from lead exposure have been established only in the past 3-4 decades.[8] Many studies have reported correlations between BLL and G.I. tract perturbations. BLLs of 50-70 µg/dL generally result in abdominal pain and other G.I. tract disturbances, while 80-100 µg/dL can cause severe colic.[37,38] One study reported radiographic classification for gastroduodenal disorders treated at the University of Naples (Italy) medical clinic form 1919 to 1958.(39) During these years a total of 148 occupationally exposed patients were admitted with BLLs over 100 µg/dL for 20% of the cases. Thirty-one of the patients were symptomatic for abdominal colic, 12 expressed symptoms for ulcer and 80 reported duodenitis symptoms. X-ray evidence was established for spastic colitis in 118 cases, gastroduodenitis in 62 patients, and duodenal ulcer for 13 cases.

The human reproductive system in not to be left out of the physiologic systems susceptible to lead intoxication. The first reports referred to women workers in lead industries at the end of the 19th century.(40) These reports outlined cases of sterility, amenorrhea and other menstrual disorders. The teratogenetic effects of lead leading to malformations, spontaneous abortion, shortened gestation and premature delivery were discussed recently by Zeilhuis.(41)

Lerda reported on the toxic effects of lead on the production and transport of sperm in men.(42) The study included 38 exposed workers with BLLs ranging from 40-98 µg/dL and 30 non-exposed controls. The exposed workers had a significant decline in sperm count (68 vs. 101 X 10^6 cells/ml) and motility (50 vs 70%), respectively.

Lead intoxication has also been shown to be causal for depressed thyroid function. Robins et al. studied 47 male workers employed by a small foundry.[43] Twenty of the men were Caucasians and 27 were Blacks. Both groups had similar mean ages and work histories. T4 levels for the entire group ranged from 3.4 to 10.7 µg/dL, however, T4 levels were significantly lower for Blacks. BLLs in Whites ranged from 16-65 µg/dL with a mean value of 40 µg/dL. For Blacks, BLLs ranged from 30-127 µg/dL with a mean of 60 µg/dL.

Inorganic lead is included in the IARC Monographs on Evaluation of Carcinogenic Risks to Humans.[44] IARC classifies inorganic lead in group 2B, citing that, although there is inadequate evidence for this rating from human data, animal data do provide sufficient evidence.

Most of the evidence directly supporting the carcinogenicity of lead in humans comes from case studies. One of the more convincing cases was reported by Baker et al.[45] This report involved a worker with 22 years of occupational exposure to inorganic lead. The worker was admitted to the hospital suffering from chronic renal insufficiency and lead poisoning (BLL > 60 µg/100g). Examination and subsequent histopathological studies indicated the presence of a cystic renal carcinoma. Chemical analysis of the tumor tissue determined that it contained 2.47 µg Pb/gram while the renal medulla contained only 0.78 µg Pb/gram. This renal carcinoma was very similar to renal carcinomas found in laboratory animals with chronic lead exposure.

A large retrospective study investigating deaths due to malignant neoplasms among 7,000 workers with exposures to lead in foundries and lead-battery factories was reported by Cooper and Gaffey.[46] A higher incidence of mortality was recorded for smelter workers due to several types of malignant tumors, however, the same finding could not be determined from data on battery plant workers. In addition, no correlation was found between the estimated level of lead exposure and carcinoma incidence.

Lead exposures are wide-spread in the workplace. As recently as 1992, Matte tabulated over 100 activities which could expose workers to a variety of lead compounds.[47] Most of these exposures result in entry via the respiratory tract. Airborne lead levels were reported for a broad range of industries in the latter 1960s.[48] High-end exposure levels for specific processes in several industries ranged from 3.88-13.10 milligrams/cubic meter (mg/m^3). This study and many others resulted in the adoption of health standards in Europe and the United States in the 1970s with enormous impact. One report in Finland illustrates the effects of this effort.[49] This study documented that mean BLL for 2,209 workers in 182 workplaces declined from 53 µg/dL in 1968 to 25 µg/dL in 1977.

Froines et al. reported airborne lead levels taken from Occupational Safety and Health Administration (OSHA) inspections conducted during 1979-1985.[50] A total of 52 "high risk industries" were identified by the authors. Those industries where more than one third of the OSHA inspections discovered airborne lead levels greater than the permissible exposure limit (PEL) were classified as high risk industries. The five high risk industries with the highest airborne lead levels were railroad equipment; bridge, tunnel, and elevated highway; highway and street construction; construction and mining machinery; and truck and bus bodies. Mean exposures measured in these industries were 10,265, 1,470, 1,005, 815 and 680 micrograms/cubic meter ($\mu g/m^3$), respectively. The OSHA Standard for lead became effective March 1, 1979.[51]

Mean airborne lead levels for the 52 high risk industries ranged from 30-10,265 $\mu g/m^3$. Clearly, hazardous levels of airborne lead were present in a large number of industries. Radiator repair shops were classified under the category of automotive repair shops with a mean airborne lead level of 95 $\mu g/m^3$.[50]

Elevated lead exposures in radiator repair shops continue to be reported by investigators.[52-56] Radiator repair workers routinely use torches to remove solder from radiators to repair leaks or to replace defective parts that cannot be patched with solder. Solder also is used to reassemble repaired parts. The solder typically used for these repairs is an alloy containing 40 percent tin and 60 percent lead. When this type of solder is used, the radiator repair process leads to the production of large quantities of lead and lead oxide fumes. Recommendations have been published concerning ventilation control of these lead exposures.[57,58] However, most shops employ fewer than 11 employees, and any given shop would seldom if ever receive on-site evaluation/inspection by regulatory agencies.[53] Thus, it is unlikely that current knowledge concerning exposures and controls reaches the individual repair shop worker.

In the State of Oregon, Oregon Health Division (OHD) initiated a lead surveillance program in 1990 that was funded by the National Institute for Occupational Safety and Health (NIOSH).[59] The OHD program requires that laboratories performing serological evaluation for lead report to OHD all test results in which the BLL is $\geq$ 25 micrograms per deciliter (µg/dl). In the first year (August, 1990-August, 1991) of the program, 100 cases were reported to OHD; 43 of the 100 cases were a result of radiator repair. Through 1994, 167 cases directly as a result of radiator repair were reported. Of the 167 cases $\geq$ 25 µg/dl, 56% had elevated blood lead levels (EBLL) between 25-39 µg/dl, while 23%, 16%, and 5% had EBLLs between 40-49 µg/dl, 50-59 µg/dl, and $\geq$ 60 µg/dl, respectively.

It was within this context that the Oregon Occupational Safety and Health Division (OR-OSHA) elected to initiate a special emphasis program focusing on radiator repair shops within the state and under OR-OSHA jurisdiction. The emphasis program focused on compliance with lead exposure regulations and hazard communication regulations.[60,61] These two Oregon codes contain the same regulations present in the federal codes plus additional, Oregon-initiated rules. Serious hazards not related to these two codes, but observed during an on-site inspection, were cited also.

METHODS

Radiator repair shops throughout the state of Oregon were identified using the 1990-91 Oregon Business Directory (compilation of the telephone yellow pages). Shops located in the nine counties that adjoin or are within the Willamette Valley were then extracted from this master list. The Willamette Valley was selected for the study because of population demographics. One hundred and one shops were listed for the nine counties. Weighting was assigned to each county based on the number of shops within that county. The initial approach was to inspect (with air monitoring) at least one shop in the county with the lowest weighting. This approach required air monitoring inspections at a total of twenty shops. Counties and number of shops selected to be inspected were Benton (1), Clackamas (3), Lane (2), Linn (1), Marion (3), Multnomah (6), Polk (1), Washington (2) and Yamhill (1).

The specific shops to be inspected were selected randomly from the total number of shops in a given county. Each shop site was visited chronologically based on the sequence of the random draw for each county. If the shop selected was non-jurisdiction (sole proprietorship) or out-of-business as determined by site visitation, an additional shop in the same county was selected by random draw. This process was continued until a sufficient number of shops had been inspected to satisfy the weighting for each county.

Inspections were conducted exclusively by the author and carried out as per Oregon Administrative Rules (OAR 437-01-055 through 437-01-251). Inspections included all general recordkeeping and reporting requirements, all relevant requirements from the Hazard Communication and Lead Standards, and serious violations of other codes if observed. Serious violations are violations wherein hazards are present that could result in serious physical harm. Air monitoring, sampling, wipe tests and all analytical laboratory procedures were conducted as per standard OSHA protocols. Air monitoring was conducted in those shops doing one or more radiator repair jobs per day. Inspections at air monitoring sites always involved at least two site visits. The first site visit included an opening conference, a site walkaround, and subsequent planning with the owner to perform airborne lead monitoring at the earliest possible date.

During each inspection, a number of items were noted. These items included the number of repair jobs conducted per day (determined by verbal questioning), ventilation details, layout of shop (with photographs), shop dimensions, personal protective equipment worn by each worker including respiratory protection used (if any), and worker work practices. Repair workers present on the day of the inspection were interviewed concerning the Hazard Communication and Lead Standards.

Repair workers also answered a voluntary questionnaire (Appendix A) about smoking history, lead health hazard training, torch fuel used, eating in the work area, personal hygiene habits throughout and at the end of the workday, work history, blood lead testing, treatment for elevated blood lead levels, symptoms potentially related to chronic lead exposure and medical treatment for clinical symptoms.

RESULTS

Fifty radiator repair shop sites (Table 1) were visited in an attempt to identify those doing one or more radiator repair jobs per day. Fourteen (28%) non-jurisdiction and nine (18%) out-of-business shops were identified during site visitation of the fifty shops, attesting to the high percentage of radiator shops operated as sole proprietorships as well as the competition level that exists in this industry. Four shops were distributor/manufacturers and did not conduct repairs. One shop was under OR-OSHA consultation at the time of the attempted compliance inspection; no inspection was conducted at this site. Emphasis program inspections were conducted at the remaining twenty-two shops. Of these twenty-two shops, only nineteen were conducting one or more radiator repair jobs per day and thus met the study criteria for conducting airborne lead monitoring. The three shops where airborne lead exposures were not measured were evaluated for compliance with all other relevant regulations. The number of radiator repair workers employed by the twenty-two shops ranged from one to five with a median of two.

The planned number of airborne lead inspections for each county was achieved except for Clackamas. Only two sites were identified in Clackamas county which met our criteria of one repair job per day. Thus, more than fifty inspections may have produced additional air monitoring sites, however, these sites would have been outside of Clackamas County. Hence, we chose to terminate the study at only nineteen air monitoring sites.

TABLE 1

Radiator Shop Site Classification

Classification	Number of Shops
OR-OSHA Inspection Conducted	22
No Jurisdiction	14
Out of Business	9[A]
Distributor/Manufacturer	4[B]
OR-OSHA Consultation	1[C]

[A] One of these shops closed shortly after the emphasis inspection was opened and remained closed throughout the emphasis program.
[B] No radiator repair operations in progress.
[C] An OR-OSHA consultation was determined to be in progress during the opening conference. No inspection was conducted.

One-hundred and fifty-one violations were cited at the 22 worksites inspected (Table 2), a mean of 6.9 violations per shop. Two shops had 14 violations, and only one shop was violation free. Of the 151 violations, 104 (mean 4.7 per shop) were classified as serious and 47 (mean of 2.1 per shop) were classified as general. Shops that had never received OR-OSHA consultation had an average of 5.6 serious and 2.4 general violations. The four shops that had received OR-OSHA consultation had an average of 0.75 serious and 0.75 general

TABLE 2

Total Violations Cited at Each Worksite Inspection Listed Chronologically

Worksite Number	Opening Date	Number of Employees[A]	Total Violations
1	1/17/91	2	8
2	4/09/91	1	6
3	4/11/91	2	2
4	4/18/91	2	5
5	4/18/91	2	4[B]
6	5/09/91	2	14
7	5/14/91	2	14[C]
8	7/25/91	2	11
9	8/22/91	1	4
10	8/28/91	2	1[D]
11	9/30/91	2	6
12	10/30/91	3	11
13	12/05/91	2	0[D]
14	1/30/92	2	11
15	2/13/92	2	13
16	3/11/92	1	9
17	3/23/92	3	11
18	3/25/92	2	13
19	4/09/92	3	2
20	4/23/92	1	1[C]
21	5/01/92	1	4[D]
22	5/28/92	5	1[C,D]

[A] This represents the number of workers that actually repair radiators. These workers may perform other tasks in addition to radiator repair.
[B] This site used lead-free solder during the monitoring portion of the inspection.
[C] These three worksites were under the same ownership.
[D] These sites had requested and received OR-OSHA consultation prior to the compliance inspection.

violations. The one shop that had no violations had received OR-OSHA consultation prior to the compliance inspection.

Airborne lead exposures (Time Weighted Average (TWA) @ 8 hr.) were measured only for workers repairing radiators on the day of inspection. Many radiator repair workers also perform administrative duties depending on daily workload. Consequently, lead exposures (Table 3) were measured for 32 of the 45 radiator repair workers and exposures ranged from 6.2-236.8 micrograms/cubic meter ($\mu g/m^3$) with a mean of 76.7 $\mu g/m^3$ (σ = 58.4). One repair worker was monitored twice for airborne lead due to a job change between inspections. Another repair worker switched to lead-free solder during the period between the opening inspection and the air monitoring inspection, resulting in laboratory results indicating no lead exposure. A follow-up airborne lead monitoring inspection for this site revealed that the repair worker had converted back to lead based solder and airborne lead exposure measured 38.6 $\mu g/m^3$.

Eight (25%) of the 32 workers had never had a BLL measurement (Table 4). Nineteen of the workers had received BLL measurements in the calendar year prior to the compliance inspection. Average BLL for these 19 workers was 34.7 $\mu g/100g$ (σ = 10.8) whole blood and ranged from 17-61 $\mu g/100g$. The highest reported BLL was the only repair worker that voluntarily admitted on our questionnaire to experiencing and having been diagnosed with clinical symptoms that could be

TABLE 3

Personal Airborne Lead Exposures[A]
At Sites Where Air Monitoring Was Performed

Worksite Number	Employee Designation	Airborne Exposure[B]
1	E-1	47.4
3	E-2	92.2
3	E-3	36.9
5	E-4	00.0[C]
6	E-5	60.9
6	E-6	54.8
7	E-7	139.9
8	E-8	51.0
8	E-9	86.6
10	E-10	45.2
11	E-11	32.6
12	E-12	45.5
12	E-13	62.0[D]
13	E-14	6.2
13	E-15	18.7
14	E-16	60.2
14	E-17	192.0
15	E-18	154.0
15	E-19	163.0
16	E-20	166.0
17	E-21	93.8
17	E-22	39.6
18	E-23	48.5[E]
18	E-24	167.0[E]
19	E-25	17.1
19	E-26	53.6
19	E-27	43.3
20	E-28	47.3
21	E-29	39.7
22	E-30	41.6
22	E-31	33.8
22	E-32	236.8

[A] Eight hour time-weighted averages.
[B] Units of micrograms of lead per cubic meter of air.
[C] This site used lead-free solder during the monitoring inspection.
[D] Employee E-13 was monitored earlier as employee E-7.
[E] These measurements are 8.5 hour time-weighted averages.

TABLE 4

Blood Lead Measurements At Sites Where Air Monitoring Was Performed

Worksite Number	Employee Designation	Date of Inspection	Date of Measurement[A]	Blood Lead[B] Measurement
1	E-1	1-17-91	2-15-88	38
3	E-2	4-11-91	3-13-91	27
3	E-3	4-11-91	8-23-90	33
5	E-4	4-18-91	6-13-90	32
6	E-5	5-09-91	7-16-90	41
6	E-6	5-09-91	7-16-90	41
7	E-7	5-14-91	2-18-91	36.9
8	E-8	7-25-91	2-17-89	43
8	E-9	7-25-91	-------	------[C]
10	E-10	8-28-91	-------	------[C]
11	E-11	9-30-91	3-20-91	34
12	E-12	10-30-91	-------	------[C]
12	E-13	10-30-91	-------	------[C,D]
13	E-14	12-05-91	2-05-91	33.9
13	E-15	12-05-91	2-05-91	30.7
14	E-16	1-30-92	-------	------[C]
14	E-17	1-30-92	-------	------[C]
15	E-18	2-13-92	-------	------[C]
15	E-19	2-13-92	-------	------[C]
16	E-20	3-11-92	-------	------[C]
17	E-21	3-23-92	3-28-91	37.7
17	E-22	3-23-92	3-19-91	18
18	E-23	3-25-92	4-16-92	50
18	E-24	3-25-92	3-23-92	61
19	E-25	4-09-92	12-9-91	53.9
19	E-26	4-09-92	12-9-91	17
19	E-27	4-09-92	12-9-91	14.2
20	E-28	4-23-92	2-13-92	34
21	E-29	5-01-92	5-01-91	24
22	E-30	5-28-92	12-13-91	35
22	E-31	5-28-92	4-14-92	35
22	E-32	5-28-92	12-13-91	39

[A]Date of most recent measurement.
[B]Units of micrograms of lead per 100 grams of whole blood.
[C]These employees had no blood lead measurements prior to inspection.
[D]This individual had been monitored for airborne lead exposure at a different worksite. Blood lead measurements form the earlier site were 36.9 μg/100g on 2-18-91.

related to chronic lead exposures. Only 11 workers had performed BLL measurements during the six months prior to inspection as required for employees who are or may be exposed to 30 $\mu g/m^3$ for 30 days per year.[60] Six workers reported BLL above 40 µg/100g. Thirteen citations were written for biological monitoring for lead, and one citation was written for failure to comply with medical removal requirements.

Of the 151 citations shown in Table 5, 85 involved violations of the Lead Standard while 44 involved violations of the Hazard Communication Standard. Four of the six most frequently cited violations were lead code violations with biological monitoring for lead and lead exposure training violations accounting for the two most frequent citations written. The remaining two most frequently cited violations of the lead code were airborne lead overexposures and lead contaminated work surfaces. At least one citation was written for each of eight additional rules related to the Lead Standard. The least commonly cited violations of the lead code were high BLL medical removal, failure to have a written compliance program, and failure to monitor after equipment modification, each cited only once. The two most frequently cited violations of the Hazard Communication Standard were failure to have a written hazard communication program and missing material safety data sheets (MSDSs). A common citation not related to these two codes was failure to post a Safety and Health Protection Poster. The most commonly

TABLE 5

Number of Citations for Each Specfic
Health and Safety Code Cited During Inspections

Code Cited	Citations
1910.1025(j)(2)(i)(A)+(B)-Biological Monitoring for Lead	13
1910.1025(l)(1)(ii)-Lead Exposure Training	13
1910.1025(c)(1)-Airborne Lead Exposure	12
1910.1025(h)(1)-Lead Contaminated Work Surfaces	12
OAR-437-155-015(1)-Written Hazard Communication Program	12
OAR-437-155-025(1)-Missing Material Safety Data Sheets	12
1910.1025(d)(2)-Initial Lead Monitoring	11
OAR-437-155-030-Hazard Communication Training	11
OAR-437-01-275(2)(a)-Safety/Health Protection Poster	10
1910.1025(m)(2)(i)-Lead Exposure Warning Signs	9
1910.1025(g)(2)(vii)-Lead Contaminated Clothing Containers	7
OAR-437-155-020(5)+(7)-Hazard Communication Container Labels	7
OAR-437-50-025(2)-Eye/Face Protection When Using Caustic	4
OAR-437-129-025(4)+(5)-Personal Respirator Maintenance	4
1910.1025(j)(3)(i)(A)-Medical Exam for Lead Exposed Worker	3
OAR-437-155-015(1)(a)-Hazard Communication Chemical List	3
OAR-437-01-705(1)-OSHA 200 Form Available at Worksite	2
1910.1025(d)(8)(i)-Employee Notification of Lead Monitoring	2
1910.1025(d)(7)-Lead Monitoring after Equipment Modification	1
1910.1025(e)(3)(i)-Overexposure Written Compliance Program	1
1910.1025(k)(1)(i)(D)-High Bloodlead Medical Removal	1
1910.215(b)(9)-Abrasive Grinder Tongue Guard	1
Total	151

cited issue outside of the two focus codes that could result in serious health consequences dealt with the failure to use splash protection when placing radiators and parts in caustic baths. Respirators were rarely in use in these shops and the few respirators found were poorly maintained.

The class of ventilation relied upon to remove soldering fumes from the work area was documented for each of the nineteen sites where airborne lead was monitored (Table 6). Nine of the sites relied entirely on general ventilation to remove fumes. Only two of the sites used a ventilated enclosure as described in a recent NIOSH paper.[58] One of the shops that used ventilated enclosures for two workers also had the lowest airborne lead exposures measured. This shop had received an OR-OSHA consultation and was the only shop in the study with no citations. The remaining shops used either a canopy receiving hood or a capture hood. One site provided a capture hood for one worker and general ventilation for a second worker. Nine of the ten shops that relied on general ventilation for at least one worker had airborne lead exposures over 50 $\mu g/m^3$ (OSHA PEL). It was observed that the same types of ventilation were utilized differently by different workers due to their different work practices.

Early in the study, wipe samples indicated that equipment/work surfaces associated with the repair of radiators in all shops were macro-contaminated with lead during the repair process. It became apparent during the two

TABLE 6

Ventilation in Use at Sites
Where Air Monitoring was Performed

Worksite Number	Lead[A] Exposure(s)	Ventilation
1	47.4	Canopy[B]
3	92.2/36.9	General[C]
5	0.0	Canopy[D]
6	60.9/54.75	Capture[E]
7	139.9	Capture
8	51.0/86.6	General
10	45.2	Enclosure[F]
11	32.6	Canopy
12	45.5/62.0	General
13	6.2/18.7	Enclosure
14	60.2/192	Capture/General[G]
15	154/163	General
16	166	General
17	93.8/39.6	General
18	48.5/167	General
19	17.1/53.6/43.3	General
20	47.3	Capture
21	39.7	General
22	41.6/33.8/236.8	Capture

[A]Units for lead exposure measurements are micrograms of airborne lead per cubic meter of air.
[B]Canopy receiving hood. This type of ventilation usually consists of a ventilated canopy hood positioned a few feet above test tank surface.
[C]No local ventilation at the immediate point where radiators are repaired. Workers must rely on general building ventilation to remove soldering fumes.
[D]Used lead-free solder at the time of the inspection.
[E]Also called a capture hood. Local ventilation that utilized flexible tubing to place intake very close to the point of generation of fumes.
[F]Soldering was performed inside a local ventilated five-sided enclosure.
[G]Worker with 60.2 $\mu g/m^3$ exposure used capture ventilation. Worker with 192 $\mu g/m^3$ exposure used general ventilation.

separate inspection days at each air-monitored worksite that these work surfaces were seldom cleaned, although contamination from contact with these surfaces was common. Only one shop had a strict policy of daily decontamination of work surfaces. Wipe samples were also taken on shop and office telephones, office desks, coffee prep areas, breakroom tables and locker room benches depending on the existence and use of these items/areas at each worksite (Table 7). All wipe samples tested positive for the presence of lead with laboratory results ranging from 9.3-402,400 micrograms/100 square centimeters ($\mu g/100cm^2$). It was especially common for repair workers to answer the telephone during the radiator repair process. One shop telephone tested 1680 $\mu g/100cm^2$ in the region normally in contact with the hand. At one worksite, a lead contaminated telephone was cleaned using only a water wet paper towel to determine if surface lead could be easily removed. A wipe sample taken immediately after cleaning tested negative for lead. Thus, lead removal is not a difficult task, but would be time consuming due to contamination of a broad spectrum of commonly used work surfaces.

During air monitoring at two of the shops, coincidental timing for the replacement of filter cassettes with new cassettes revealed that short-term exposures to a specific operation could result in elevated airborne lead exposure. One worker was observed to be exposed for a period of sixty

TABLE 7

Surface Lead Contamination

Worksite Number	Wipe Sample Location	Quantity of Lead Present[A]
1	Test Tank	402,400
2	Test Tank	110,000
3	---------	-------[B]
4	Test Tank	123,500
5	Test Tank	22,910
6	Walking Surface	Bulk Solder[C]
7	Shop Telephone	36.2
8	Shop Telephone	1680
9	Work Surface	Bulk Solder[C]
10	Shop Telephone	19.2
11	Office Telephone	22.9
12	Shop Telephone	14.0
13	Shop Telephone	60.4
14	Office Desk	690.0
15	Coffee Table	442.0
16	Shop Telephone	9.3[D]
17	Coffee Prep Area	69.0[E]
18	Shop Telephone	341.0
19	Break Room Table	23.8
20	Locker Room Bench	84.0
21	Telephone Desk	81.6
22	Break Room Table	38.6

[A]These values represent the micrograms of lead removed by wiping approximately 100 square centimeters of surface.
[B]All work surfaces and floors cleaned each day at end of shift.
[C]A bulk sample was taken from the repairman walking/working surface.
[D]The surface of this phone was cleaned using a water wet paper towel after initial wipe sample was taken. A wipe sample taken after cleaning the phone showed no detectible lead.
[E]Samples taken from shop telephone (117μg) and picnic table (78.6μg).

minutes to average airborne lead concentration of 1,060 $\mu g/m^3$. The operation performed during this sixty minute period was a tear-down involving core header cleaning, commonly called a "header job". On a separate occasion at a different shop, a worker was observer to be exposed to 7.0 $\mu g/m^3$ for 228 minutes doing routine patch and boltup work followed by 479 $\mu g/m^3$ for 234 minutes performing the same routine work with the exception of approximately 45 minutes spent cleaning the core headers of an industrial radiator. Thus, it appears that core header cleaning can be responsible for macro-overexposures.

Each radiator repairman was asked to complete a voluntary questionnaire (Appendix A). Using this questionnaire, the average age and average number of years employed as a radiator repairman for this group of 32 workers were determined to be 38 years and 15.3 years, respectively. Only one of the workers was female. Fifteen of the 32 workers reported the use of some type of respirator (usually a half-mask cartridge respirator) at least occasionally. However, the author observed the use of respirators for only two of the workers. In this instance, the shop owner required that respirators be used at all times during soldering or solder removal. Six of the workers reported experiencing symptoms selected from the voluntary questionnaire. As discussed earlier, only one of these workers had been diagnosed by a physician with symptoms characteristic of chronic lead poisoning.

DISCUSSION

Results of this study are in agreement with earlier investigators regarding the potential for radiator repair workers to be exposed to airborne lead fumes and consequently to develop elevated blood lead levels (EBLLs).[52,53,55,58] Limited evidence also supports recommendations for the use of ventilated enclosures ;[58] however, because of the compliance nature of our study, no attempt was made to investigate the effectiveness of any given class of ventilation on lead fume exposures.

No attempt was made to correlate BLL with airborne lead exposures. However, airborne and topical lead exposure potentially leading to EBLLs appears to be largely a consequence of worker work practices. This parameter is difficult to quantify in radiator repair shops. However, a given worker's work practices are a consequence of that worker's selection of: 1) the various techniques requiring different quantities of lead solder that can be utilized for the same repair process; 2) worker production pace; and 3) possible approaches for utilization of existing ventilation.

Thus, worker practices can vary dramatically from worker to worker at the same shop while performing the same repair process utilizing the same ventilation. However, the potential for large variation in worker work practices does not imply that installation of local ventilation is not necessary to reduce airborne and topical exposures in shops where only general ventilation is present or existing local ventilation can be improved.

Clearly, access to properly designed local ventilation is fundamental to reducing exposures.[58] However, training workers regarding good work practices resulting in minimizing the use of lead solder has the potential to minimize release of lead fumes thereby reducing the quantities of fumes that must be removed by existing ventilation. Good work practices also can result in the capture of lead fumes nearer to the source, thus reducing the release of fumes to the general environment of the shop.

An unusual aspect of our study is the context of compliance within which the investigation was conducted. The shops were given no advance notice of impending inspection/evaluation unlike previous studies.[52,53,58] Inspected shops and workers could not decline airborne lead monitoring as reported in earlier work.[52,53] Because the inspections were carried out under the auspices of OR-OSHA, all previous airborne and biological monitoring data for air-monitored workers were available. Each shop was evaluated for compliance with all aspects of the Lead Standard and Hazard Communication Standard. In addition to airborne lead and biological monitoring, shops were evaluated from the standpoint of lead exposure training, contaminated work surfaces, material safety data sheets for lead solder, lead exposure warning signs, lead contaminated clothing containers, respirator maintenance, medical exams for lead exposed workers, and high blood lead medical removal as well as all aspects of hazardous chemical exposure regulations. Consequently, a relatively complete picture of health and safety practices was determined for each worksite. The number of violations however, may have been influenced somewhat by the chronological sequence of inspections; that is, owners of shops inspected late in the program may have learned of the increased likelihood of inspection and taken actions to reduce the number of rule violations. The compliance context also could have contributed to perturbations in worker behavior and repair workload on the day of airborne monitoring for lead in some instances; both of these factors, if present, would have resulted in reduced airborne lead exposures.

CONCLUSIONS

It is apparent that most radiator repair shop owners of shops located in the Willamette Valley in western Oregon lack the knowledge or means to institute established practices useful in reducing exposure to lead. Lack of preventative practices is evident from the large number of violations of the Lead Standard at the 22 inspected shops (an average of slightly less than four per shop). In addition, four of the six most frequently cited violations were violations of the lead code, and nine of the ten shops using general ventilation were cited for personal airborne lead exposures above the permissible exposure limit (PEL).

Two of the four most frequently cited violations of the lead code were airborne lead overexposures and lead contaminated work surfaces. With a mean of 76.7 $\mu g/m^3$ for all air monitored shops, and seven workers and six shops with airborne exposures more than double the PEL and one exposure of 236.8 $\mu g/m^3$, it clear that high airborne lead exposure levels are possible in these shops. It is likely that "header jobs" are responsible for a large portion of the airborne lead associated with many of these high airborne lead exposures. It also appears that airborne lead exposures can be substantially reduced through the use of ventilated enclosures.

It is difficult to assess the association between topical exposures and chronic lead poisoning. The potential for topical exposures in the inspected shops is high because a broad spectrum of wipe tested surfaces was contaminated with lead. Movement of lead/lead oxide fumes to work surfaces is likely due to the colloidal size of the particles generated and likelihood of transfer via workers hands and clothing. The high potential for topical exposure points to the need for rigid personal hygiene practices, routine cleaning of surfaces contacted by all workers, and restriction of repair workers to the repair area unless workers decontaminate.

The use of OR-OSHA consultants results in compliance with standards and, more importantly, results in reduced airborne lead exposures. The value of OR-OSHA consultants is apparent because only one of the seven repair workers in shops that received OR-OSHA consultation prior to our inspection was determined to be overexposed as measured by air monitoring. In addition, these same four shops received only three citations (less than one per shop) from the Lead Standard during compliance inspections. Shops that have access to OSHA or worker's compensation consultants can benefit from compliance with the Lead Standard.

CONCLUDING REMARKS

OSHA and OHD have successfully developed regulations that address the risks associated with lead usage in radiator shops.[59,60] In addition, NIOSH has published technology appropriate for reducing lead exposures in these workplaces.[58] However, several factors inherent to radiator shops not peculiar to our study act as barriers to the goal of reduced lead exposures.

The foremost barrier is economics. Based on estimates from NIOSH,[58] the installation of appropriate ventilation can cost between $1000 - $1500 per worker. This estimate does not include maintenance, operation and increased HVAC costs associated with local ventilation. Due to the size of the average radiator shop, this capital cost could represent a significant financial commitment that does not result in additional income. The proper operation and use of local ventilation also may require changes in established habits resulting in perceived as well as real inconveniences. Thus, the installation of appropriate ventilation equipment is likely to be viewed as an expensive, non-income producing burden.

Other barriers include knowledge and training levels for shop owners and workers. The primary focus of the shop owner is the solvency of their business. Knowledge and training would be totally palatable only when leading to this end.

Since most exposures in radiator repair shops lead to BLLs that produce sub-clinical symptoms, the linkage between financial success and lead exposure in not clearly evident. Consequently, regulatory concern (OSHA) is the primary motivating force to cause workers and owners to focus on lead exposures. OSHA rules have limited impact in workplaces unless owners and workers understand and are concerned about taking steps to reduce risk. The reality of most radiator repair shops seems to be that state or federal regulatory intervention to reduce lead exposures is viewed as unneeded intrusion.

In the state of Oregon, regulatory agencies (OSHA and OHD) should strive to create more "user-friendly" images, focusing more on positive, results-oriented outcomes and less on strict rule compliance. Radiator trade associations should act as motivating forces to encourage shop owners to develop a clear understanding of the risks associated with radiator repair work. Encouragement should be presented in the context of worker health and shop viability as opposed to fear of OSHA or other regulatory agencies. More specifically, regulatory agencies, trade associations or possibly large individual radiator repair shops should clearly identify those portions (i.e., header jobs, etc.) of the radiator repair process that represent the greatest risk and then develop simple guidelines to reduce this risk.

RECOMMENDATIONS

A logical extension of this study would be to investigate airborne lead exposures more systematically, statistically comparing different types of repair jobs performed by the same repair person. Of even greater interest would be comparisons between different repair workers performing the same repair job using the same work station and ventilation.

BIBLIOGRAPHY

1. Hamilton, A. and Hardy, H.L.: Industrial Toxicology. 3rd ed. Publishing Sciences Group, Inc., Boston, Mass. (1974).

2. Nriagu, J.O.: Lead and Lead Poisoning in Antiquity. John Wiley and Sons, New York (1983).

3. Nicander of Colophon, cited in: Matte D.T., Landrigan, P.J. and Baker, E.L.: Occupational Lead Exposure. In Needleman H.L. (Ed.). Human Lead Exposure CRC Press Boca Raton, Florida (1991).

4. Bisel, S.C.: Int. Health Magazine. 5:32 (1983).

5. Gilfillan, S.C.: Lead Poisoning and the Fall of Rome. J. Occup. Med. 7:63-68 (1965).

6. Crushiny, A.R.: Textbook of Pharmacology and Therapeutics. Churchill, London (1910).

7. Gould, S.E.; Kullmann, N.J. and Sheckett, H.A.: Effect of Lead Therapy on Blood Cell of Cancer Patients. Am. J. Med. Sci. 194:304 (1937).

8. Castellina, N., Castellino, P., and Sammolo, N.: Inorganic Lead Exposure. Lewis, Ann Arbor (1995).

9. Rabinowitz, M.B.,: Toxicokinetics of Bone Lead. Environ. Health Perspect. 91:33-37 (1991).

10. Barry, P.S.I.: A Comparsion of Concentrations of Lead in Human Tissues. Br. J. Ind. Med. 32:119-139 (1975).

11. Barry, P.S.I. and Mossman,D.B.: Lead Concentrations in Human Tissues. Br. J. Ind. Med. 27:337-351 (1970).

12. Alessio,L. and Foa, V.:Lead. In: Alessio L., Berlin, A., Roi, R. and Boni, M. (Eds.), Human Biological Monitoring of Industrial Chemical Series. C.E.C. Industrial Health and Safety. EUR 8476 EW. pp. 115-117 (1983).

13. Mahaffey, K.R., Annest, J.L., Roberts, J., and Murphy, R.S.: National Estimates of Blood Lead Levels: United States, 1976-1980. Association with Selected Demographic and Socioeconomic Factors. New Engl. J. Med. 370:573-579 (1982).

14. Fribergh, L. and Vahter, M.: Assessment of Exposure to Lead and Cadmium Through Biological Monitoring: Results of a UNEP/WHO Global Study. Environ. Res. 30:95-128 (1983).

15. Roberts J., Mahaffey, K.R. and Annest, J.L.: Blood Lead Levels in General Populations. In: Mahaffey K. (Ed.), Dietary and Environmental Lead: Human Health Effects. Elsevier, Amsterdam. pp.355-372 (1985).

16. Quinn M. J.: Factors Affecting Blood Lead Concentrations in U.K.: Results of the EEC Blood Lead Surveys, 1979-1981. Int. J. Epidemiol. 14(3):420-431 (1985).

17. Alessio, L. and Dell' Orto, A.: Treatment of Metal Intoxication with Chelating Agents. Med. Lav. 77:639-648 (1986).

18. Araki, S., Aono, H., Fukahoi, M. and Tabuki, K.: Behavior of Lead and Zinc in Plasma, Erythrocytes, and Urine and ALAD in Erythrocytes Following Intravenous Infusion of CaEDTA in Lead Workers. Arch. Environ. Health. 39:363-367 (1984).

19. Martell, A. E. and Calvin, M.: Chemistry of the Metal Chelated Compounds. Prentice-Hall, Englewood Cliffs, N.J. Append-1 (1952).

20. Foreman, H. and Trujillo, T.T.: The Metabolism of C^{14} Labelled Ethylenediaminetetraacetic Acid in Human Beings. J. Lab. Clin. Med. 43:566-71 (1954).

21. Remy, P.: Encephalopathie et Calcifications Cerebelleuses du Saturnisme. Presse. Med. 19:997-1000 (1990).

22. Ryan, C.M., Morrow, L., Parkinson, D., and Bromet, E.: Low Level Lead Exposure and Neuropsychological Functioning in Blue Collar Males. Int. J. Neurosci. 36:29-39 (1987).

23. Lacereaux, E.: Note Relative a un Cas de Paralyse Saturnine Avec Alteration des Cordons Nerveux des Muscles Paralyses. Gaz. Med. Paris 709-713.

24. Campbell, B. C., Beattie, A. D., Moore, M. R., Goldberg, A. and Reid, A. G.: Renal Insufficiency Associated with Excessive Lead Exposure. Br. Med. J. 1:482-485 (1977).

25. Chisolm, J. J., Harrison, H. C., Eberlein, W. R., and Harrison, H. E.: Aminoaciduria, Hypophosphatemia and Rickets in Lead Poisoning. Am. J. Dis. Child. 89:159-165 (1955).

26. Cooper, W. C., Wong, O., and Kheifets, L.: Mortality Among Employees of Lead Battery Plants and Lead Producing Plants. 1947-1980. Scand. J. Work Environ. Health. 11:331-345 (1985).

27. Garrod, A. B.: The Nature and Treatment of Gout and Rheumatism. Walton and Maberly, London, pp. 260-273 (1895).

28. Lancereaux, E.: Nephrite et Arthrite Saturnines Coincidence de ces Affections; Parallile Avecla Nephrite et L'arthrite Gouttenses. Arch. Gen. Med. Dec:641-653 (1881).

29. Morgan, J. M., Harteley, M. W. and Miller, R. E.: Nephropathy in Chronic Lead Poisoning. Arch. Intern. Med. 118:17-29 (1966).

30. Laennec, R. T. H.: Traite' de l' Auscultation Mediate. Chaude'. Paris. p. 79 (1831).

31. Aub, J. C.: Lead Studies III. The Effects of Lead in Red Blood Cells. Part 3. A Chemical Explanation of the Reaction of Lead with Red Blood Cell. J. Exp. Med. 40:189-288 (1924).

32. Watson, C. J.: J. Clin. Invest. 15:327-340 (1936).

33. Whitehead, J.: The Establishment and Interpretation of Dose-Effect Relationships for Heavy Metal. Monitoring and Assessment Research Center Technical Report. 18 (1980).

34. Baldi, G. and Sbertoli, C.: Evoluziane delle Alterazioni Renali che Possono Condurre al Rene Grinzo Saturnino. Med Lav. 48:553 (1957).

35. Ramirez-Cervantes, B. et al.: Health Assessment of Employees with Different Body Burdens of Lead. J. Occup. Med. 20:610-617 (1978).

36. Kirkby, H. and Gyntelberg, F.: Blood Pressure and Other Cardiovascular Risk Factors of Long-Term Exposure to Lead. Scand. J. Work Environ. Health. 11:15-19 (1985).

37. Beritic, T.: Lead Concentration Found in Human Blood in Association with Lead Colic. Arch. Environ. Health. 23:289-291 (1971).

38. Dahlgren, J.: Abdominal Pain in Lead Workers. Arch Environ. Health. 21:156-159 (1978).

39. Mungo, A. and Sessa, G.: Quadro Radiologico Dell' apparato Digerente nel Saturnismo Professionale. Minerva Gastroenterol. 6(4):163-171 (1960).

40. Gerber, G. B., Leonard, A., and Jaequet, P.: Toxicity, Mutagenicity and Teratogenicity of Lead. Mutat. Res. 76:115-141 (1980).

41. Zeilhuis, R. L.: Biological Exposure Limits: The Fetus and EEC Politics. Br. J. Ind. Med. 42:145-146 (1985).

42. Lerda, D.: Study of Sperm Characteristics in Persons Occupationally Exposed to Lead. Am. J. Ind. Med. 22:567-571 (1992).

43. Robins, J. M. et al.: Depressed Thyroid Indexes Associated with Occupational Exposure in Inorganic Lead. Arch. Int. Med. 143:220-224 (1983).

44. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Supplement 7. IARC, Lyon, France (1987).

45. Baker, E. L. et al.: Occupational Lead Exposure, Nephropathy and Renal Cancer. Am. J. Ind. Med. 1:139-148 (1980).

46. Cooper, W. C. and Gaffey, W. R.: Mortality of Lead Workers. J. Occup. Med. 17:100-107 (1975).

47. Matte, D. T., Landrigan, P. J., and Baker, E. L.: Occupational Lead Exposure. In: Needleman H. L. (Ed.), Human Lead Exposure. CRC Press., Baco Raton, FL (1992).

48. Horiuchi, K., Osaka City Med. J. 16:1-28 (1970).

49. Hemberg, S. and Tola, S.: The Battle Against Occupational Lead Poisoning in Finland. Experiences During the 15 Year Period 1964-1978. Scand. J. Work Environ. Health. 5:336-344 (1979).

50. Froines, R. J. et al.: Am. J. Ind. Med. 18:1-17 (1990).

51. Occupational Safety and Health Administration: 29 CFR 1910.1025. Federal Register, 44:53007 (November 14, 1978).

52. Goldman, R.H.; Baker, E.L. ; Hannan, M., et al.: Lead Poisoning in Automobile Radiator Mechanics. N. Engl. J. Med. 317:214-218 (1987).

53. Lussenhop, D.H.; Parker, D.L.; Barklind, A.; et al.: Lead Exposure and Radiator Repair Work. Am. J. Public Health 79(11): 1558-1560 (1989).

54. Froines, J.R.; Baron, S.; Wegman, D.H.; et al.: Characterization of the Airborne Concentrations of Lead in U.S. Industry. Am. J. Ind. Med. 18:1-17 (1990).

55. Papenak, P.J.; Ward, C.E.; Gilbert, K.M.; et al.: Occupational Lead Exposure in Los Angeles County. Am. J. Ind. Med. 21:199-208 (1992).

56. Bellows, J.; Rudolph, L.: The Initial Impact of a Workplace Lead-Poisoning Prevention Project. Am. J. Public Health 83(3):406-410 (March 1993).

57. Centers for Disease Control and Prevention. Control of Excessive Lead Exposure in Radiator Repair Workers. MMWR. 40: 139-41 (1991).

58. Goldfield, J.; Sheehy, J.W.; Gunter, B.J.; et al.: Cost Effective Radiator Repair Ventilation Control. Appl Occup. Environ. Hyg. 6(11): 959-965 (1991).

59. Oregon Health Division, Office of Epidemiology & Health Statistics: Current Disease Summary--Surveillance for Elevated Blood Lead Levels. Volume 40, No. 8 (April 9, 1991).

60. Oregon Occupational Safety and Health Division: Oregon Administrative Rules. Chap. 437. Division 2. General Occupational Safety and Health Rules. Subdivision Z: Lead (1910.1025). Amendments through 11/16/90.

61. Oregon Occupational Safety and Health Division: Oregon Administrative Rules. Chap. 437. Division 155. Hazard Communication. Amendments through 2/8/88.

APPENDIX

Appendix A

SPECIAL EMPHASIS LEAD PROJECT
INTERVIEW QUESTIONNAIRE

1. Name ______________________________

2. Date ____/____/____

3. Interviewer ______________________________

4. Employer ______________________________

5. Address ______________________________

6. Telephone Number ______________________

7. Occupational Title ______________________________

8. Current Employment Information

   A. Years with current employer. ____________ (years)

   B. Describe your current job. ______________________________
   ____
   ____
   ____

9. Age ____________

10. Sex Male _____ Female _____

11. Race White _____ Hispanic _____
    Black _____ Indian _____
    Asian _____ Other _____

12. Have you ever smoked cigarettes? Yes _____ No _____
    (No means less than 20 packs in a lifetime.)

13. If yes to 12. What is your smoking history? ____________
    ____
    ____

14. Have you received any training on the health hazards of lead? If yes, describe training. Yes _____ No _____
    Training? ______________________________
    ____
    ____
    ____
    ____

15. Are you familiar with Material Safety Date Sheets (MSDS)? Yes _____ No _____

16. Have you read the MSDS for lead? Yes _____ No _____

17. What fuel is used for the torch while using lead based solder. Natural Gas ____ Acetylene ____ Other ____

18. Do you use a respirator while soldering? Yes ____ No ____ Sometimes __________ (%)

19. Describe your respirator. How is it maintained? ________
____
____
____

20. Have you ever been fit tested for respirator usage? Yes ___ No ___ When was the last time you were fit tested? ___________

21. Do you eat in the work area? Yes _____ No _____

22. How often do you wash your hands while at work (Times/Day)? _______________

23. Do you shower after work? Yes _____ No _____

24. What protective clothing do you wear while soldering? Gloves _____ Long Sleeves _____ Hat _____

25. Do you change clothes at the end of shift? Yes __ No __

26. Describe the mode of ventilation used at your soldering work station. ______________________________
____
____
____
____
____

27. Describe the types of past employers, your job titles, and duration of employment for previous employment.

Employer Type	Job Title	Duration

28. Have medical tests been performed on you in the past to determine level of lead in blood or urine? Yes ___ No __

29. If yes, what were the results? ______________________________
____
____
____
____
____
____

30. Name and address of physician that performed lead tests.
____
____
____
____
____

31. Have you ever undergone treatment for elevated lead levels? Yes _____ No _____

Describe treatment. ______________________________
____
____
____
____

32. Since the beginning of your current employment, have you experienced any of the following symptoms?

	Yes	No
A. General		
Weight Loss	______	______
Fatigue	______	______
Decreased Appetite	______	______
B. Head, Eyes, Ears, Nose		
Headaches	______	______
Decreased Visual Activity	______	______
Hearing Deficits	______	______
Ringing in Ears	______	______
Metallic Taste in Mouth	______	______
C. Cardiopulmonary		
Shortness of Breath	______	______
Chronic Cough	______	______
Chest Pains	______	______
Palpitations	______	______

	Yes	No
D. Gastrointestinal		
Nausea	___	___
Vomiting	___	___
Heartburn	___	___
Abdominal Pain	___	___
Constipation	___	___
Diarrhea	___	___
E. Neurologic		
Irritability	___	___
Insomnia	___	___
Dizziness	___	___
Loss of Memory	___	___
Confusion	___	___
Hallucinations	___	___
Incoordination	___	___
Decreased Strength (Hands/Feet)	___	___
Disturbances in Gait	___	___
Difficulty in Climbing Stairs	___	___
Seizures	___	___
F. Hematologic		
Paleness	___	___
Abnormal Blood Loss	___	___
Black Stool or Vomit	___	___
G. Reproductive		
History of Infertility	___	___
Impotence	___	___
History of Miscarriages, Stillbirths	___	___
H. Musculoskeletal		
Muscle and Joint Pains	___	___

33. Have you seen a physician for any of the above symptoms?
Yes ______ No ______

34. If you have seen a physician for any of the above items, may I have permission to request your medical record?
Yes ______ No ______

Name and address of physician. ______________________________
____
____
____